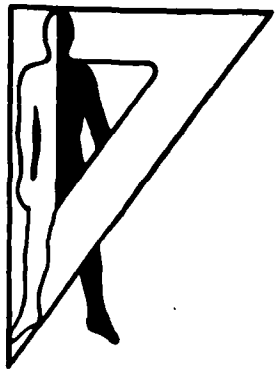

Technical Memorandum 3-81

IMPLICATIONS OF A CRITICAL LEVEL IN THE EAR FOR ASSESSMENT NOISE HAZARD AT HIGH INTENSITIES

G. Richard Price

February 1981
AMCMS Code 612716.H700011

U. S. ARMY HUMAN ENGINEERING LABORATORY
Aberdeen Proving Ground, Maryland

Destroy this report when no longer needed.
Do not return it to the originator.

The findings in this report are not to be construed as an official Department of the Army position unless so designated by other authorized documents.

Use of trade names in this report does not constitute an official endorsement or approval of the use of such commercial products.

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER: Technical Memorandum 3-81	2. GOVT ACCESSION NO.: AD-A097 204
3. RECIPIENT'S CATALOG NUMBER:	
4. TITLE (and Subtitle): IMPLICATIONS OF A CRITICAL LEVEL IN THE EAR FOR ASSESSMENT OF NOISE HAZARD AT HIGH INTENSITIES.	5. TYPE OF REPORT & PERIOD COVERED: Final
	6. PERFORMING ORG. REPORT NUMBER:
7. AUTHOR(s): G. Richard Price	8. CONTRACT OR GRANT NUMBER(s):
9. PERFORMING ORGANIZATION NAME AND ADDRESS: U.S. Army Human Engineering Laboratory, Aberdeen Proving Ground, MD 21005	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: AMCMS Code 611716.H700011
11. CONTROLLING OFFICE NAME AND ADDRESS:	12. REPORT DATE: February 1981
	13. NUMBER OF PAGES: 8
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office):	15. SECURITY CLASS. (of this report): Unclassified
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE:

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Noise
Noise Hazard
Damage-Risk Criterion
Critical Level for the Ear
Exposure to Noise
Stapes Displacements

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The argument is advanced that there is a critical level (CL) for the ear at high intensities where the loss mechanism undergoes a fundamental change. In physiological terms, CL probably represents the intensity at which a given level of mechanical stress is reached in the Organ of Corti. Electrophysiological, histological, and threshold shift data support this contention. Further, CL varies as a function of the spectrum of the sound.

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

A preliminary estimate of the freefield SPL reaching CL in the human ear has been calculated as has been the distribution of CL. The implications for human exposure and damage-risk criteria are discussed.

AMCMS Code 612716.H700011

Technical Memorandum 3-81

IMPLICATIONS OF A CRITICAL LEVEL IN THE EAR FOR ASSESSMENT NOISE HAZARD AT HIGH INTENSITIES

G. Richard Price

February 1981

APPROVED: [signature]
JOHN D. WEISZ
Director
U.S. Army Human Engineering Laboratory

U.S. ARMY HUMAN ENGINEERING LABORATORY
Aberdeen Proving Ground, MD 21005

Implications of a critical level in the ear for assessment of noise hazard at high intensities

G. Richard Price

US Army Human Engineering Laboratory, Aberdeen Proving Ground, Maryland 21005
(Received 18 February 1980; accepted for publication 23 September 1980)

The argument is advanced that there is a critical level (CL) for the ear at high intensities where the loss mechanism undergoes a fundamental change. In physiological terms, CL probably represents the intensity at which a given level of mechanical stress is reached in the Organ of Corti. Electrophysiological, histological, and threshold shift data support this contention. Further, CL varies as a function of the spectrum of the sound. A preliminary estimate of the freefield SPL reaching CL in the human ear has been calculated as has been the distribution of CL. The implications for human exposure and damage-risk criteria are discussed.

PACS numbers: 43.50.Qp, 43.66.Sr

INTRODUCTION

"I'm worried by . . . reviving the ghost of the critical intensity. I thought maybe we had gotten rid of that thing for all time. I say this because if there is a critical intensity, we are going to have even more problems trying to make a single damage risk standard that encompasses exposures from 75 to 80 dB up to impulse noises of 160 dB or so." (W. D. Ward[1]).

A number of lines of evidence converge to argue that there is indeed a critical level (CL) for the ear at high intensities, above which the mechanism of loss is fundamentally different from that at lower intensities. Exactly what CL means in physiological terms remains to be established, although indications are pointed out later in the article. The important point, so far as assessment of noise hazard is concerned, is that the mechanism of loss above CL is such that the ear is likely to suffer permanent changes even from very short exposures. Consequently, exposures above CL should be considered extremely hazardous and avoided. There is also evidence that CL is dependent on the spectral distribution of the energy. The implication of the foregoing, as Ward pointed out, is that a damage-risk criterion (DRC) for exposure to noise that is useful below CL will not predict well above it; consequently, more than one form of DRC will be required to cover noise hazards ranging from typewriters to cannons. It follows that proposals using the same predictive scheme from 60 or 70 dB up to as high as 180 dB (Atherley and Martin[2]; EPA[3]; Johnson[4]; and Martin[5]) should be re-examined.

The primary function of this paper is heuristic. Evidence for the presence of a critical level will be presented, and the implications for the rating of hazard will be discussed. The evidence comes from a variety of sources, some of it from the research of scientists who may not themselves have viewed the data as demonstrating the presence of a critical level. As might be expected, the data are not yet definitive, however, they *are* consistent with the theoretical position that there is a spectrally dependent CL in the ear. If additional research does confirm the presence of CL, then damage-risk formulations will have to recognize its presence, and instruments designed to measure hazard will have to accommodate it.

Two further general comments are in order before the main arguments are made. First, the reader will notice that the traditional distinctions between continuous, impulse, and impact noises are not made. This is deliberate. In thinking about the ear's response to intense sounds, it may be more profitable to focus on the absolute level of the sounds rather than on the mechanism by which the sounds are produced. This focus sharpens the view with respect to the mechanism of loss operating within the ear and makes the assessment of hazard somewhat simpler. Secondly, the mammalian ear is viewed as *similar* in design across species. This view is amply supported by years of research and is generally accepted. On the other hand, there are species differences in tuning, susceptibility to damage, etc. When processes are compared across species in this article, all that is required is that the ears be similar. When levels are calculated for the human ear, only data derived from the human are used.

I. THE EXISTENCE OF CL

A. Evidence from TTS studies

The exploratory work of Ward, *et al.*,[6] on impulsive stimulation can be interpreted as demonstrating the presence of CL. In Fig. 1, drawn from their data, we see the TTS resulting from a constant number of impulses as the intensity is raised. Clearly, the ear begins to accumulate TTS rapidly once some level is exceeded. This pattern is repeated for most of the ears they provide individual data for, although the specific level varies from subject to subject. (Presumably all ears have a CL, however, the range of intensities tested may simply have not been great enough to demonstrate it in all ears. An estimate of the variability of CL calculated later in this article is consistent with this contention.)

Additional data from this same study can be interpreted as showing that the primary mechanism producing loss of sensitivity changes at the same time. Ward *et al.*,[6] stimulated a number of ears just above CL and found that the growth of TTS was proportional to the number of impulses rather than to the logarithm of the

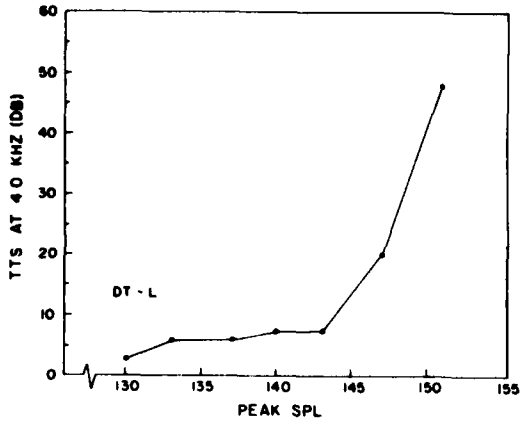

FIG. 1. Threshold shift at 4.0 kHz for one ear following increasingly intense exposure to 25 impulses in 1 min. Data for ear DT–L from Ward *et al.*[6]

number, as one would have expected from studies done with sounds at lower intensities (Ward[7]). Additionally, McRobert and Ward,[8] with essentially the same stimulating conditions, demonstrated that the equal energy principle did not apply at levels above CL. That is, the equal energy principle predicts that if the intensity were dropped 3 dB and the number of impulses doubled, the TTS would be the same. Their data showed that when the individual ears were stimulated above the critical level, TTS grew rapidly. When stimulated below it, TTS was small and roughly constant, supporting an equal energy principle below CL.

If the mechanism responsible for loss does change as a function of the level of exposure, then we might expect to find that recovery patterns change as well. Luz and Hodge[9] have focused on recovery following intense exposures, reviewing data for both human and animal ears. Recovery from lower intensity exposures normally follows a time course that is linear in log time; but following intense exposures recovery may be delayed, TTS may *increase* following exposure, recovery may be diphasic, or under certain conditions, thresholds may even show a transient increase in sensitivity ("negative TTS"). Luz and Hodge hypothesized that there are two processes responsible for loss, one of them metabolically based and the other a function of structural changes. They felt that the structural changes were produced by the most intense exposures (impulses with peak pressures from 155 to 168 dB). Their arguments and data are therefore consistent with the hypothesized presence of a critical level at high intensities.

B. Evidence from electrophysiological studies

Two lines of evidence from animal experiments fit very nicely into the argument for the presence of CL. In the first, Price[10,11] measured shifts in cochlear microphonic (CM) sensitivity in the cat ear following progressively more intense pure tone exposures. Below a certain level, losses in CM sensitivity accumulated at a rate proportional to the logarithm of the exposure time; however, at a higher level, the rate of loss increased sharply and became directly proportional to the exposure time. A second line of evidence appeared in a later series of experiments in which cat ears were exposed to spectrally narrow impulses (Price[12]). At about the same SPL as in the previous experiments, nerve potentials (N1) recorded from the round window, also began to show long lasting losses (no recovery in tests 3 h post exposure). Furthermore, the level at which these losses began to grow varied systematically as a function of the spectral location of the stimulating impulse. In a discussion of this study, these data in conjunction with data from other studies concerned with intracochlear mechanics, were interpreted as indicating that a critical level for these impulses was reached when the mechanical stress (in contrast to metabolic demand) had reached a given level at particular locations within the Organ of Corti. Expressed in terms of stapes displacements in the cat ear, this level declined at 5.4 dB/octave with increasing frequency. Assuming that a similar process operates in the human ear, the free-field SPL reaching this threshold was calculated for the human ear, and the curve indicating the relative CL as a function of frequency is reproduced here in Fig. 2. This curve predicts that CL is lowest at 3.0 kHz and progressively higher with either higher or lower frequencies. Most of the shape of this curve is due to the transfer functions of the external and midle ears, which are reasonably well known for the human ear. Consequently, if the value of 5.4 dB/octave (derived from the cat ear) should not be exactly the same for the human, it would not affect the shape of the curve in Fig. 2 very much.

C. Evidence from histological studies

Perhaps the most persuasive evidence for the presence of CL comes from histological studies of noise exposed ears. Spoendlin[13] has examined structural changes in the guinea pig ear and has found that, for a white noise, there was a critical intensity at approximately 130 dB SPL above which direct and immediate alterations in the ear's structure occurred regularly, and below which primary structural damage was excep-

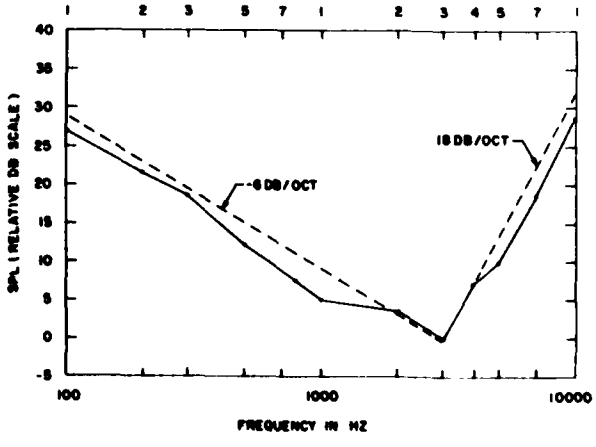

FIG. 2. Free field SPLs reaching the loss threshold for spectrally narrow tone pips as calculated for the human ear. Taken from Price.[12]

tional. Below the critical intensity, the type of changes he observed led him to conclude that the damage mechanism was primarily metabolic. As would be expected, there are some uncertainties remaining in the transition zone between a region of loss that is primarily a function of metabolic exhaustion and one of direct mechanical damage. A cell being vibrated to the limit of mechanical destruction is doubtless also in metabolic extremity and vice versa, i.e., a cell in metabolic distress may also lack its normal mechanical strength. But, in any event, it appears that a transition in mode of damage does occur with increases in intensity.

Dancer *et al.*,[14] have recently done an experiment that can be interpreted as supporting this same viewpoint. Two groups of guinea pig ears were exposed to the same energy in a 2-h period. One received a continuous noise exposure at 120 dB SPL (a one octave band centered between 4.5 and 9.0 kHz) and the other group received the energy (the same spectrum) in 10-ms gated bursts (10 ms on/4 s off) at 146 dB SPL. The group exposed to continuous noise at 120 dB showed almost no loss of hair cells within the cochlea, whereas the group exposed at 146 dB showed a total loss of hair cells in the lower two turns of the cochlea. The difference between these groups of ears is dramatic and must be based on something other than the energy delivered. If CL exists for the guinea pig ear somewhere above 130 dB, then the results make sense.

D. Summary

In summary, the foregoing arguments from a wide range of sources converge to support the following points:

(1) There is a CL for the ear at high intensities, where the rate of loss goes from being proportional to the logarithm of the exposure time to being directly proportional to the exposure time. Losses grow much more rapidly above the CL than below it, and may show complex recovery functions. In physiological terms, the CL may be the intensity at which the primary damage mechanism changes from one of metabolic exhaustion to one of mechanical stress.

(2) The specific sound pressure reaching CL varies as a function of the spectrum of the sound as shown in Fig. 2. Sounds in the vicinity of 3.0 kHz should be the most hazardous.

II. APPLYING THE CONCEPT OF CL

A. Establishing the absolute level

1. Calculation from TTS data

In order to discuss the concept of CL with an eye toward applying it to real situations, it is necessary that the ordinate on Fig. 2 have an absolute reference so that CL can be fixed. Fortunately, Ward *et al.*,[6] plotted losses for individual subjects in several figures; consequently, CL can be estimated from their data. In order to avoid the complication of middle ear muscle activity, the only data used were for the slowest pulse repetition rate (25 clicks per minute) and for which no reflex eliciting tone had been present. (The 2.4 s interpulse interval presumably allowed any elicited middle ear muscle contractions to relax before the next pulse arrived.) The data meeting these restrictions were for a total of 14 ears from 11 individuals.[15] The CL assigned each ear was the SPL above which the loss curve showed the greatest rate of increase in TTS. For instance, CL for the ear in Fig. 1 would be 143 dB. Three ears did not show the sharp increase in growth of TTS characteristic of CL before they had reached the limit of the ability of the speaker system to produce impulses (155 dB). For statistical purposes, these ears were assigned a CL of 155 dB. The mean CL was 142 dB, which is almost the same as the median CL of 141.5 dB. This mean value is probably best interpreted as representative of a young normal human population.

2. Amplitude calibration

There is some uncertainty (in addition to the normal problems of calibration) associated with the specification of peak pressures used by Ward *et al.*,[6] and McRobert and Ward.[8] The pressure reported was that of the condensation peak, even though the rarefaction peak was higher. Which peak should be used, the rarefaction or the condensation peak? At very high intensities, there could be some asymmetry in the middle ear system, i.e., the eardrum could be pulled outward further than it could be pushed inward, or the incudostapedial joint would tend to transmit strong condensations but pull apart with rarefactions. Price[16] has calculated that the annular ligament of the stapes could begin to introduce nonlinearity in the human ear for sinusoidal stimuli at these pressures by limiting stapes displacements, but the question of symmetry is still open. Some of the best measurements of middle ear function have been made on the cat ear by Guinan and Peake[17] who reported that, for frequencies below 1500 Hz, the motion of the stapes became nonlinear at about 140 dB. Furthermore, the motion was asymmetrical. The stapes moved further during the rarefaction phase than during the condensation phase. Based on this evidence, it is reasonable to suspect that for intense impulses, the rarefaction phase may be even more important than the condensation phase. The ultimate question, of course, is how such motions are related to damage, but until additional evidence is produced, a conservative approach would rate the rarefaction phase as being at least as hazardous as the condensation phase.

The exact value of the rarefaction peak is somewhat in doubt. Ward *et al.*,[6] state that the rarefaction peak was 2 dB higher than the condensation peak and, if one measures the waveform they present in their Fig. 2, this is confirmed. McRobert and Ward[8] used the same stimulating system and made the same statement about pressure, however, when the waveform they published is measured, the rarefaction peak is somewhat more than 4 dB greater than the condensation peak. The logical conclusion of these arguments is that the peak level should probably be considered as being 2 to 4 dB higher than reported in the articles. The number used here was 3 dB, and future references to pressures incorporate this correction factor. The best estimate of CL for

the impulse used by Ward *et al.*,[6] is 145 dB, once the 3-dB correction factor is applied.

3. Spectrum

The specification of CL must also include spectrum, as well as peak pressure. This impulse was a heavily damped sinusoid that had been produced by ringing a speaker with an electrical impulse. It had a predominant frequency of about 1.0 kHz, which was apparent from inspection of the published waveform. This spectrum was verified by copying the waveform into a computer and calculating the relative pressure spectral density. This result appears in Fig. 3.

4. Conclusion

We can therefore conclude from the Ward *et al.*,[6] data that, for the average ear, CL is 145 dB SPL for a damped 1.0-kHz sinusoidal impulse arriving at normal incidence to the ear. Given this one point, the curve in Fig. 2 has been fixed on an absolute scale and has been redrawn in Fig. 4 with the ordinate having absolute values.

B. Estimate of variability

If an estimate of CL is to be made for portions of the population, then it is necessary to have some estimate of the variability of CL. The standard deviation calculated from the data of Ward *et al.*,[6] is 8.5 dB, and if it is corrected to account for the small sample size, it becomes 8.8 dB. There are some additional data in the article by McRobert and Ward[8] that should be examined in this context. McRobert and Ward defined the critical exposure for their purposes as the SPL producing a 20 dB TTS at 4.0 kHz, measured 30 s after exposure to 20 impulses in 1 min. By requiring a relatively large TTS in response to a few impulses, it is to be expected that this definition will result in a somewhat higher estimate than that established earlier for CL. However, a standard deviation can be calculated for these data as a second estimate of variability.[18] The standard deviation for these data is 4.8 dB and following correction for sample size, is 5.0 dB. The discrepancy in the two estimates of variability is probably not a serious problem.

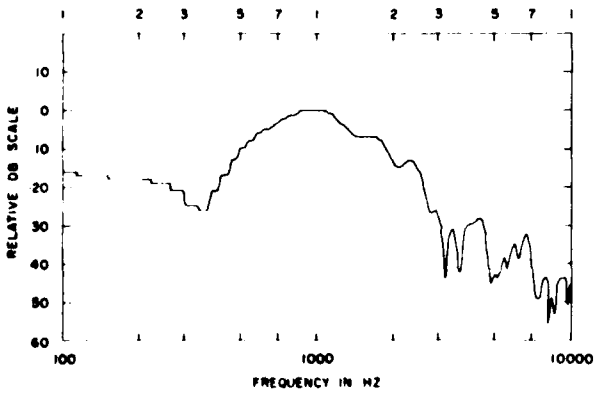

FIG. 3. Spectrum of impulse from Altec 20801 speaker used by by Ward *et al.*[6]

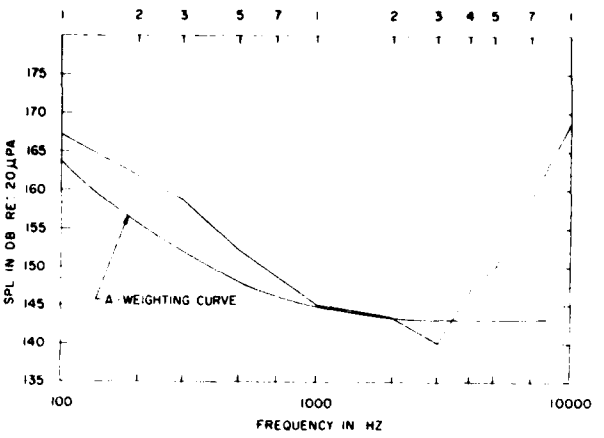

FIG. 4. CL as a function of frequency for the human ear for a damped sinusoid arriving at normal incidence. The A-weighting curve is plotted for a comparison of general shape, the location with respect to the ordinate is arbitrary.

Ward *et al.*,[6] state that the data in their figures were chosen to demonstrate both extreme and typical examples of the data they saw. It is reasonable to suppose that such a bias may have produced a slightly greater proportion of extremes than would be found in a random sampling from a normal population. Consequently, the variance of such data may be a little high (although the mean or median could be in essentially the right place.) As a concession to this possibility and to be consistent with the observation (Ward *et al.*[6]) that CL was observed at about 133 dB for one ear and exceeded 158 dB for some, a standard deviation of 8.0 dB is proposed as a reasonable estimate of the variability of CL in a normal population.

Given an 8.0 dB standard deviation and a mean CL of 145 dB, it can be calculated that CL for this impulse would be 132 dB for a 5th percentile ear (95% less susceptible) or 140 dB for a 25th percentile ear. This same procedure calculates CL for the 95th percentile ear to be 158 dB, which is consistent with the observation that 158 dB was insufficient to produce sizeable shifts in a few ears. The curve in Fig. 2 also indicates that impulses with peak energy at 3.0 kHz would be even more hazardous by 5 or 6 dB. This may explain at least part of the great efficiency of the toy noisemaker ("cricket") used once by Ward *et al.*,[6] in producing TTS. Inspection of the waveform they published for the cricket shows that it had a resonance at 3.0 kHz, which is where the prediction says the ear should be most susceptible.

C. Using the CL concept

1. Levels above CL

If CL exists, it is apparent that no DRC based on a single set of assumptions about the ear's behavior at high intensities, such as the equal energy principle, will be able to encompass the entire range of sounds to which people are exposed. For a variety of reasons, such a criterion would be desirable, but it must fail to assess the risk accurately once CL is exceeded. Con-

sequently, the efforts in this direction by Atherley and Martin,[2] Coles and Rice,[19] the United States Environmental Protection Agency,[3] and Martin,[5] need to be reexamined when CL is exceeded. A criterion for use above CL incorporating the concept of spectral distribution of energy is presently being developed (Price[12,20,21]). It is not in final form yet; however, it may be of interest to note that it differs markedly from the two DRCs in use in the world for impulse noises (CHABA[22]; Pfander[23]).

2. Determining that CL is not exceeded

A practical question is how one can determine the CL is *not* exceeded and that DRCs applicable at lower levels may be safely used. This sounds like a simple question but is in reality very complicated. It will be recalled that the most likely physiological basis for CL is that a certain level of mechanical stress has been reached within the Organ of Corti (Price[12]). Consequently, a method is needed for predicting basilar membrane displacements resulting from particular acoustic signals. Mathematical modeling efforts are presently under way in this laboratory with this ultimate goal in mind (Kalb[24]).

a. Sinusoidal signals. In the case of a sinusoidal signal, the problem of determining when CL has been reached is not very great because the curve for Cl in Fig. 4 can be used. In view of its usefulness in rating noise hazard at lower intensities, the A-weighting curve has been included in Fig. 4 for comparison. For frequencies between 100 and 3000 Hz, it is apparent that A-weighting does not do too badly at rating the risk. Below 100 Hz (not plotted in Fig. 4), the A-weighting probably cuts off a little too sharply. For instance, if an A-weighted level of 140 dB were allowable, then the attenuation imposed by A-weighting would allow pressures of over 100 psi at 10 Hz, which is obviously intolerable. There has been little systematic study of auditory hazards in the low-frequency region, however, a recent study by Burdick *et al.*,[25] indicates that low frequencies can have surprising effects. Specifically, chinchillas exposed to an octave band of noise centered at 63 Hz showed high-frequency hearing losses (in the 1.0 to 2.0-kHz region). It would be imprudent to transfer this information directly into estimates for the human ear, however, it does emphasize the lack of knowledge with respect to the effects of low-frequency stimulation and, for the purposes of the present discussion, advises caution in estimating the hazard from acoustic transients with peak energies at low frequencies.

One factor ameliorating some of the concern in the frequency region below 1000 Hz is that the curve in Fig. 4 does not make allowances for the protective effects of the middle ear muscles. Depending on a complex set of circumstances, the middle ear muscles can provide a significant but variable degree of protection primarily below 1000 Hz (Fletcher[26]; Reger[27]). Therefore the curve in Fig. 4 ought to be considered a worst-case example.

Above 3000 Hz, the A-weighting and CL curves diverge sharply. If there were signals above 3000 Hz, they would be overrated by the A-weighting network. At a practical level, this might be an insignificant problem because most noise sources have most of their energy at lower frequencies. If an error were made by using A-weighting with a sound with a great deal of high frequency energy, it would at least be in the conservative direction.

b. Complex waveforms. Almost no sounds outside the laboratory are sinusoidal, however. Given a complex waveform, it is apparent that the traditional methods of measurement do not do exactly what is required. Peak levels can be determined from a pressure-time history or from peak-reading meters, but these methods ignore spectrum. Spectral analysis, on the other hand, typically ignores phase data, and consequently, magnitude data cannot be used from such an analysis to predict instantaneous displacements within the cochlea. The heart of the problem lies in the dual quality of the auditory system as Tonndorf[28,29] pointed out some years ago. It has properties appropriate to both the time and frequency domains. One proposal has been made for rating hazard based on pressure-time data. Martin[5] and Martin and Atherley[30] have proposed a method to calculate L_{eq} for impulsive noises from the pressure-time history. Unfortunately, the method simply adds a single correction factor rather than actually A-weighting the energy. Atherley and Martin[2] have argued that this procedure adequately characterizes industrial noises, which tend to have most of their energy in the mid-frequency region. However, for sounds with most of their energy in the low-frequency region, e.g., cannon fire, this procedure would probably overrate the hazard. The traditional methods of measurement thus cannot be used for general evaluations, and the mathematical model that will explain it all and tell just what must be done has not been developed. Hopefully, once the specific needs for a measurement system have been determined, the instrument makers will be able to produce an appropriate device.

3. Possible applications

a. Sinusoidal impulses. Given that the general-purpose instrument is yet to be designed, there are still some things that can be done today. If a resonant system is driven by an impulse, then the acoustic result is essentially a damped sinusoid. A simple instance of this sort would be a speaker being driven with a step function as Ward *et al.*,[6] did in their experiment. Inspection of the pressure-time history for such a transient allows both the fundamental period and amplitude to be established reasonably well (which was how the estimate of 145 dB for CL was made in this paper).

b. Weapons impulses. For the type of transient produced by gunfire or explosions, the problem has an additional complexity. Such impulses are characterized by an essentially instantaneous rise to the peak pressure, a rapid decay through ambient pressure, a slower rise in the negative direction, usually to about 1/3 the positive peak, and a final return to ambient pressure. In spectral terms, the initial peak can be considered as being composed of many high frequencies all in phase at

the beginning of the impulse. A variety of mechanisms act in the ear to effectively attenuate this high-frequency energy, which is why the curve for CL in Fig. 4 rises sharply above 3.0 kHz. It follows that somewhat higher peak pressures can be tolerated for impulses of this type when the peak is measured from a pressure-time history. The exact amounts need to be established through additional research, however, some data indicate that peak pressures about 8 dB higher might be tolerable on this account (Price[30]). In contrast to the uncertainty in establishing a peak level, it is possible to establish the location of a spectral peak fairly easily for a "classic" weapons impulse from what is commonly termed the A-duration (the time required for the initial peak to fall to ambient pressure). It can be shown that where P is the spectral peak in Hz and

$$P = 0.279/A,$$

A is the A-duration in seconds. The fact that almost all weapons-type impulses are similar, may make it possible to rate their hazardousness relative to one another by using the general form for their spectrum and the curve in Fig. 4. As mentioned earlier, this work was begun several years ago and is approaching a final form for this type of impulse. Almost all gunfire impulses (with the possible exceptions of cap pistols and 0.22 cal weapons) have peak pressure at the firer's ear that exceed 155 dB and could, therefore, be above CL for some ears. Until a DRC for weapons' noises is formalized and tested, a conservative course of action would be to presume that they may exceed CL for at least some ears.

c. General procedures. It is apparent that there is presently no simple way to measure all waveforms to determine whether or not CL has been exceeded. By using various combinations of measurements, depending on the source of the acoustic transients, we have seen that it may be possible to make a reasonable estimate, however. *Both* peak level and spectrum need to be accounted for. What may be needed is a device that measures peak levels within 1/3 octave bands and then applies a frequency weighting to the results. For frequencies between 100 and 10 000 Hz, the weighting is probably known fairly closely, but for lower frequencies, it is not. The commercial production of a measurement system would be an important step toward making the measurement of CL practical.

III. GENERAL DISCUSSION

In order to maintain a proper perspective, it should be acknowledged at this point that the foregoing analysis of the properties of CL has been based on a limited set of data for relatively simple impulses produced in the laboratory. Such a procedure is consistent with the process of coming to grips with fundamental mechanisms producing loss in the ear, however, the real world promises to add considerable complexity to the picture. For example, Hamernik and Henderson[31] and Henderson and Hamernik[32] have documented many instances where seemingly trivial details of the exposure conditions have produced interactions that have resulted in major differences in effect. Considerable research will still need to be done to clarify such interactions.

Once CL has been exceeded, it has been argued that the ear begins to suffer from the effects of the mechanical stresses produced within the Organ of Corti. This will be reflected as a change in threshold and, if not too severe, will be temporary. There are many questions with respect to the adequacy of threshold shifts to characterize the state of well being of the ear, and no data can be produced here to reduce this concern. However, the argument that threshold shifts above CL are due to mechanical stress, coupled with the unusual patterns of recovery noted earlier, *does* suggest that threshold shifts at these levels should not be considered equivalent to threshold shifts at lower levels. At this point, there is insufficient research to say just what threshold shifts would be acceptable, but the erratic recoveries and the finding that there can be losses at the cellular level within the Organ of Corti and no permanent threshold shift (Bohne, *et al.*[33]), combine to suggest extreme caution in declaring any threshold shift produced by sounds above CL as tolerable.

[1]W. D. Ward, comment in discussion of paper in *Effects of Noise on Hearing*, edited by D. H. Henderson, R. P. Hamernik, D. S. Dosanjh, and J. H. Mills (Raven, New York, 1976) p. 89.

[2]G. R. C. Atherly and A. M. Martin, "Equivalent-continuous noise level as a measure of injury from impact and impulse noise," Ann. Occup. Hyg. **14**, 11–23 (1971).

[3]EPA, "Information on levels of environmental noise requisite to protect public health and welfare with an adequate margin of safety," Rep. 550/9–74–004, Office of Noise Abatement and Control, US Environmental Protection Agency, Washington, DC (1974).

[4]D. L. Johnson, "Some thoughts on hearing conservation: Possible corrections to equal energy role to account for intermittency and impulse noise," Aerospace Medical Research Lab, Wright–Patterson AFB, OH, AMRL-TR-75–4 (1975).

[5]A. Martin, "The equal energy concept applied to impulse noise," in *Effects of Noise on Hearing*, edited by D. H. Henderson, R. P. Hamernik, D. S. Dosanjh, and J. H. Mills (Raven, New York, 1976).

[6]W. D. Ward, W. Selters, and A. Glorig, "Exploratory studies on temporary threshold shift from impulses," J. Acoust. Soc. Am. **33**, 781–793 (1961).

[7]W. D. Ward, "Adaptation and fatigue," in *Modern Developments in Audiology*, edited by J. Jerger (Academic, New York, 1973).

[8]H. McRobert and W. D. Ward, "Damage-risk criteria: The trading relation between intensity and the number of nonreverberant impulses," J. Acoust. Soc. Am. **53**, 1297–1300 (1973).

[9]G. A. Luz and D. C. Hodge, "Recovery from impulse-noise induced TTS in monkeys and men: A descriptive model," J. Acoust. Soc. Am. **49**, 1770–1777 (1971).

[10]G. R. Price, "Functional changes in the ear produced by high-intensity sound. I. 5.0-kHz stimulation," J. Acoust. Soc. Am. **44**, 1541–1545 (1968).

[11]G. R. Price, "Functional changes in the ear produced by high-intensity sound. II. 500-Hz stimulation," J. Acoust. Soc. Am. **51**, 552–558 (1972).

[12]G. R. Price, "Loss of auditory sensitivity following exposure to spectrally narrow impulses," J. Acoust. Soc. Am. **66**, 456–465 (1979).

[13]H. Spoendlin, "Anatomical changes following various noise exposures," in *Effects of Noise on Hearing*, edited by D. H. Henderson, R. P. Hamernik, D. S. Dosanjh, and J. H. Mills (Raven, New York, 1976).
[14]A. Dancer, R. Franke, D. Lombardo, and R. Pujol, "Effets lesionnels des bruits sur la cochlee du cobaye, etude histologique," Institut Franco–Allemand de Recherches de Saint-Louis, Rapport R103/80, Saint-Louis, France (1980).
[15]The values for CL assigned to specific ears were: DF–R: 140 dB DF–L: 147 dB DT–R: 133 dB DT–L: 143 dB MB–R: 139 dB MB–L: 143 dB VA–R: 130 dB RK–R: 133 dB KN–L: 139 dB DW–L: 133 dB GL–L: 143 dB JS–R: > 155 dB RS–R: > 155 dB HH–L: > 155 dB.
[16]G. R. Price, "Upper limit to stapes displacement: Implications for hearing loss," J. Acoust. Soc. Am. **56**, 195–197 (1974).
[17]J. J. Guinan, Jr., and W. T. Peake, "Middle-ear characteristics of anesthetized cats," J. Acoust. Soc. Am. **41**, 1237–1261 (1967).
[18]McRobert and Ward[8] report that the critical level, as they defined it, was 146 dB for two ears, 152 dB for four ears, and 155 dB for one ear. Four further subjects were tested. For two subjects, two minutes at 155 dB just reached the critical level. An estimate of critical level for these ears was set at 157.5 dB. For two additional ears, even two minutes exposure at the maximum did not reach the critical level.
[19]R. R. A. Coles and C. G. Rice, "Towards a criterion for impulse noise in industry," Ann. Occup. Hyg. **13**, 43–50 (1970).
[20]G. R. Price, "Spectrum as a factor in rating the hazard from intense acoustic transients," J. Acoust. Soc. Am. Suppl. **1,65**, S30 (1979).
[21]G. R. Price, "Toward a theoretically based DRC for impulse noise," J. Acoust. Soc. Am. Suppl. **1,62**, S95 (1977).
[22]CHABA, "Proposed damage-risk criterion for impulse noise (gunfire)," Report of Working Group 57, NAS–NRC Committee on Hearing, Bioacoustics and Biomechanics, Washington, DC (1968).
[23]F. Pfander, *Das Knalltrauma* (Springer–Verlag, New York, 1975).
[24]J. T. Kalb, "Response of a linear-network ear model to impulsive stimuli," J. Acoust. Soc. Am. Suppl. **1,65**, S30 (1979).
[25]C. K. Burdick, J. H. Patterson, B. T. Mozo, and R. T. Camp Jr., "Threshold shifts in chinchillas exposed to octave bands of noise centered at 63 and 100 Hz for three days," J. Acoust. Soc. Am. **64**, 458–466 (1978).
[26]J. L. Fletcher, "Protection from high intensities of impulse noise by way of preceding noise and click stimuli," J. Aud. Res. **5**, 145–150 (1965).
[27]S. N. Reger, "Effect of middle ear muscle action on certain psychophysical measurements," Ann. Otol., Rhinol., Laryngol. **69**, 1179–1198 (1960).
[28]J. Tonndorf, "The response of cochlear models to aperiodic signals and to random noise," J. Acoust. Soc. Am. **32**, 1344–1356 (1960).
[29]J. Tonndorf, "Time/frequency analysis along the partition of cochlear models: A modified place concept," J. Acoust. Soc. Am. **34**, 1337–1350 (1962).
[30]A. M. Martin and G. R. C. Atherley, "A method for the assessment of impact noise with respect to injury to hearing," Ann. Occup. Hyg. **16**, 19–26 (1973).
[31]R. P. Hamernik and D. Henderson, "The potentiation of noise by other ototraumatic agents," in *Effects of Noise on Hearing*, edited by D. H. Henderson, R. P. Hamernik, D. S. Dosanjh, and J. H. Mills (Raven, New York, 1976).
[32]D. Henderson and R. P. Hamernik, "Impulse noise induced hearing loss: An overview," in *Noise and Audiology*, edited by D. M. Lipscomb (University Park, Baltimore, 1978).
[33]B. Bohne, D. H. Eldredge, and J. Mills, "Cochlear potentials and electron microscopy applied to the study of small cochlear lesions," Ann. Otol. **82**, 595–608 (1973).